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Hidradenitis suppurativa after renal transplantation: Complete remission after switching from oral cyclosporine to oral tacrolimus

To the Editor: Hidradenitis suppurativa (HS) is a chronic, recurrent inflammatory dermatosis involving apocrine gland–bearing skin, whose management has no formal guidelines.[1]

Fig 1. Hidradenitis suppurativa. Facial nodular acne and inflammatory subcutaneous nodules on the axillae appeared in the setting of renal transplantation while the patient was on cyclosporine.

Fig 2. Hidradenitis suppurativa. All facial and axillary inflammatory lesions were cleared in a few months on tacrolimus, leaving unsightly scars.

In November 2010, a 23-year-old Indian man consulted for severe facial acne associated with suppurating lesions of major body folds, which had started 2 years after he underwent kidney transplantation in 2007 for renal failure of unknown origin. His immunosuppressive treatment included cyclosporine (Cs) 150 mg/day and mycophenolate mofetil 1.5 g/day. Treatment with isotretinoin (20 mg/day) was introduced in April 2010 but led to worsening of facial nodular acne. Subsequently, inflammatory subcutaneous nodules developed on

the buttocks, axillae, and groins and evolved in crops. Besides these nodules, physical examination revealed bridged scars, but no interconnecting tracts. Histologic examination of a skin biopsy and cultures ruled out Crohn disease and an opportunistic infection, respectively. The diagnosis was HS grade II according to the Hurley classification. A 6-month course of oxacillin (2 g/day), followed by a combination of rifampicin (600 mg/day) with clindamycin (600 mg/day) for 9 months were ineffective (Fig 1). In February 2012, Cs was replaced with tacrolimus (2 mg/day) based on the hypothesis of a deleterious effect of Cs on pilosebaceous apparatus and of a potential efficacy of tacrolimus as suggested in another report.[2] In November 2012, all inflammatory lesions had disappeared (Fig 2).

To our knowledge, this patient is the first case of HS appearing in the setting of organ transplantation. Although a fortuitous association cannot be excluded, considering the patient's age, the clinical scenario speaks in favor of an iatrogenic origin of HS. Indeed, rare cases of iatrogenic HS have been reported.[3] Even if several Cs-responsive cases of HS exist,[4] Cs is known to induce hyperplasia of the pilosebaceous apparatus.[5] Isotretinoin may also have played an aggravating role. This patient is similar to one suffering from HS for more than 20 years, in whom treatment with Cs had been ineffective.[2] He was cured at the age of 40 years, a few months after kidney transplantation, while receiving tacrolimus and mycophenolate mofetil as immunosuppressive treatment. We recently observed another case of HS that appeared within 2 years after transplantation, for which we suggested a switch from Cs to tacrolimus. Although tacrolimus and Cs exert very similar immunosuppressive effects, the former proved more effective than the latter against HS in these cases. This may be due to the fact that tacrolimus exerts fewer effects on the pilosebaceous apparatus.

This observation suggests that patients in whom HS develops while on Cs should be switched to tacrolimus.

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Ipilimumab-associated Sweet syndrome in a melanoma patient

To the Editor: We read with interest the recently published letter "Ipilimumab-associated Sweet syndrome in a patient with high-risk melanoma,"[1] in that we had a patient with a very similar presentation. A 57-year-old man with stage IIIC (T4b, N3, cM0) melanoma was enrolled in a phase 3 randomized study of adjuvant ipilimumab. Eleven days after his first infusion of ipilimumab, he presented to his oncologist with an itchy rash. He was on no other new medications and denied fever, diarrhea, myalgias, or other systemic symptoms.

The patient subsequently presented to the dermatology service 6 weeks after his first dose of ipilimumab with widespread erythematous, edematous papules and plaques, some with pseudovesicular appearance, and scattered pustules on the face, trunk, arms, and dorsal hands (Fig 1). At the time of evaluation, there was no evidence of melanoma progression. Complete blood count was within normal limits without peripheral neutrophilia. Skin biopsies were performed and demonstrated prominent papillary dermal edema with extensive neutrophilic infiltrate extending to the subcutaneous tissue with scattered eosinophils and plasma cells (Fig 2). The histopathologic features were consistent with Sweet syndrome (SS). The patient was treated with oral prednisone, initial dose of 60 mg daily, with rapid improvement. Prednisone was subsequently